

NEW YORK NEURULOGICAL SOCIETY.

Stated Meeting, April 6th, '97. B. Sachs, M.D., President.

THE STATE OF THE REFLEXES IN SUPRA-LUMBAR LESION OF THE CORD.

Dr. Joseph Fraenkel read this paper. He said that it was a general clinical rule that interference with the conduction of the spinal cord leads to an increase in the reflex activity of the centres in the cord below the lesion. The following illustrative cases were cited:

Case 1. A patient of sixty years entered the Montefiore Home fourteen months after the beginning of the disease. During the first six months he suffered from sharp, darting pains in the lower extremities. Extensive bed-sores developed during the last four months of life. The examination showed total motor and sensory paraplegia. There was no tenderness over the spinous process, and no localized atrophy. The autopsy revealed caries of the bodies of the eighth and ninth dorsal vertebræ, with great thickening of the dura at this level, and adhesion to the vertebræ for about three inches. The cord was markedly softened, and the microscope showed extensive changes, mainly of the nature of myelomalacia. Above the lesion there was secondary degeneration of the columns of Goll; below the lesion secondary degeneration of the pyramidal tracts. The process was probably tubercular.

Case 2. A woman, twenty-one years of age, entered the hospital on account of transverse myelitis. The disease had developed without assignable cause, nine months previously, but she had not sought relief until the involvement of the bladder. Six months after the appearance of the first symptom she was completely paraplegic. The examination showed marked anæmia; temperature 102° F.; extensive bed-sore over sacrum; no atrophy; lower extremities decidedly tender and flexed at the hip and knee. There was total motor and sensory paralysis of the lower

extremities. All reflex activity in the lower extremity was lost, yet, on tickling the soles of the feet, they were drawn up. The autopsy showed, at the level of the eighth dorsal vertebræ, a fistula leading into the spinal canal, and a very vascular sarcoma, the size of a cherry, which had pressed upon and softened the tenth and eleventh dorsal segments. The microscope showed that some healthy fibres kept up the continuity of the cord.

Case 3. The patient was forty years of age, and had a good personal and family history. Up to two weeks before admission his disease showed the familiar symptoms of marked pulmonary tuberculosis. At this time the spinal cord became involved in the process. The examination showed the temperature to be 101° F.; there was an extensive tubercular process in each lung; the spinal cord was tender from the lower dorsal region downward; he was greatly emaciated, but there was no localized atrophy. The gait was uncertain, apparently due to imperfect co-ordination. There were numerous areas of anæsthesia and analgesia. All superficial reflexes were present, but somewhat diminished. Fairly good contraction of the quadriceps tendon could be *felt* on tapping the tendon, but it evoked no locomotion. Five days after admission, motor paralysis was found to be more extensive, and the bladder was beginning to be involved. The patellar reflex was much diminished. Shortly after this the patellar reflex disappeared altogether, and a bed-sore developed. The post-mortem showed advanced tuberculosis in both lungs, and a pachymeningitis extending from the seventh to the tenth dorsal segments. The cord was very much softened in this region, and the microscope showed a fair number of healthy fibres. The lumbar cord was normal.

Case 4. This was a boy whose trouble had developed immediately after a severe fall. On admission the extremities presented a peculiar swollen appearance, and pitted slightly on deep pressure. There was a large bed-sore over the sacrum. The temperature was 103° ; respirations were abdominal; urine contained considerable albumen; pupils were unequal; abdomen very tympanitic; hyperæsthesia in upper part of trunk, and total anæsthesia below. The temperature sense was perverted. The muscles were in a state of complete flaccidity. There was total motor and sensory paralysis. When the soles were tickled the limbs were drawn up. At the autopsy, the

sixth, seventh and eighth dorsal segments were found to be replaced by a fibrous band.

The foregoing cases, Dr. Fraenkel said, showed that occasionally lesions of the cord above the lumbar region did not cause loss of the reflexes. He had found ninety new cases reported in the literature, representing a variety of pathological conditions, distributed in widely different parts of the cord. In the old cases there was absolute loss of motility. When the continuity of the cord is completely interrupted, this can be easily understood, but not so when the interruption is only partial. It seemed reasonable to infer that the motor fibres were always the first ones to suffer in a pathological process. Sensibility was absolutely lost in all cases where section of the cord was complete. The fibres that serve to conduct the deep sensibility seemed to have a more specialized function, and were probably the ones most prone to suffer next to the motor fibres when the cord was subjected to pressure. In nearly every case it was stated that the sphincters were paralyzed. In fifty-one reported cases, and in one of his own, there was total destruction of the continuity of the cord, and in all the lesions were such as to indicate serious injury to the cord. In his opinion Bastian's theory seemed to have met with the most general acceptance. The exact nature of the knee phenomenon was not definitely known. We know positively that the influence exerted by a higher centre could inhibit the reflex, and it seemed reasonable that this superior influence should run in an arc. The centripetal branch of this arc was in some of the ascending columns of the cord to which no definite function had yet been allotted. It should be remembered that loss of the knee-jerk in supra-lumbar lesion was not necessarily a matter of importance in prognosis.

Dr. M. Allen Starr said that he had just been reading a paper covering somewhat the same ground—a paper by Kocher, of Berne. He reported seventy cases of transverse lesion of the cord, and made very positive statements to the effect that loss of the knee-jerk is absolutely diagnostic of total abolition of the continuity of the cord above the lumbar region. Many neurologists had doubted the validity of that proposition, and hence the paper just presented was particularly timely. Bastian's statements had provoked criticism from many quarters. This paper admitted the possibility of a transverse lesion in some cases causing exaggeration, and in other

cases a loss of reflexes. While this might not satisfy our theories, it was an important contribution to the facts.

Dr. Onuf said that Dr. Fraenkel had called attention to the rôle played by the cerebellum in the production of the knee-jerk. We knew further that the pyramidal tracts had an inhibitory influence on the knee-jerk. He desired to advance a theory in this connection. We had not been able to explain many physiological facts except by assuming the influence of so-called inhibition. One important example of this was to be found in the pyramidal tracts, particularly in those fibres passing from the cortex directly to the anterior horn cell. So far as he knew, no one had tried to supply an anatomical basis for inhibition in order to explain the occurrence of excitation in one case, and inhibition in another. Dr. Onuf said that he assumed that when a current from a cell passed from the protoplasmic process towards the nervous process, or from the cell body to the nervous process, excitation of the cell resulted. If, on the contrary, the current passed from the axis cylinder or nervous process towards the cell body, inhibition was the result. These connections were not entirely theoretical—they had been actually demonstrated. He also assumed that the axis cylinder of the pyramidal fibre ended with the terminations around the axis cylinder of the anterior horn cell. The pyramidal fibres, he said, had not only an inhibitory influence on the knee-jerk, but their function was, in general, not a motor one, but an inhibitory one. He would say that voluntary excitation took place through the cerebellum, and that the direct cortical spinal fibres had only an inhibiting influence. A case of absolute paralysis in connection with degeneration of the pyramidal tract had never been reported. By assuming a motor pathway, this inhibition would be found very important in finely graduating the movements. We knew nothing definite about the mode of termination of the fibres around the peripheral neuron. According to the theory just propounded, a complete transverse lesion, interrupting the pyramidal tracts, should cause loss of the knee-jerks. Interruption of the motor cerebellar spinal pathway should cause complete loss of the knee-jerk, for the peripheral stimulus is counteracted by the inhibitory influence of the pyramids. One fact agreed very well with this theory, viz.: the degeneration of cortical spinal fibres (pyramidal tracts) frequently associated with degeneration of the anterior horn cells.

Dr. C. L. Dana said that Dr. Onuf's theory was very ingenious, but it was open to criticism, which, however, should not be made until after further consideration of it. The views of Dr. Fraenkel seemed to him very similar to those of Dr. Onuf, and they were certainly very plausible. The chief problem presented was to explain those cases in which the knee-

jerks were absent, and yet the transverse division of the cord is not complete.

Dr. Fisher asked Dr. Onuf if the pyramidal tracts were no longer to be considered as motor tracts, but rather as simply inhibitory tracts.

Dr. Onuf replied that the pyramidal fibres were not the motor tracts in the sense in which we had hitherto considered them. There must be at least this motor pathway, although there might be another one, through the pons, for example. When we stimulated the cortex, the fibre which gives the motion is the one which passes into the pons and cerebellum and into the cord.

Dr. Fisher asked why, then, with absolute destruction of the cerebellum, as had been done experimentally, we did not get absolute paralysis.

Dr. Onuf replied that *all* of the cerebellum had not been extirpated. He did not claim that only motor pathway was through the cerebellum, but that there must be another motor pathway than the pyramidal tract.

Dr. C. A. Herter said that the cases reported by Dr. Fraenkel were very valuable. His experiences certainly showed that it was possible to have a complete abolition of the patellar reflex with a partial transverse lesion of the cord above the knee-jerk centre. His own impression, gained from clinical observation and from a perusal of the literature, had been that we were justified in regarding a loss of the knee-jerks, in consequence of a transverse lesion high up in the cord, as an evidence that the transverse lesion was a complete one. He was now disposed to think that we had perhaps been rather hasty in considering this a final conclusion. While he had some hesitancy in throwing aside the motor function of the pyramidal tract, he believed the theory presented by Dr. Onuf embodied certain valuable suggestions.

Dr. Joseph Collins said that he had been particularly gratified with the paper, because, when a few months ago he had ventured the statement before the Society that in cases of partial lesion of the cord there was sometimes absence of the knee-jerk, he had been told that this must be an error of observation. But the theory that Dr. Fraenkel had proposed concerning the reflexes presented serious anatomical difficulties. While the theory that the fibre conducts impulses in the direction in which it degenerates was no longer tenable, it seemed to him that, as a general rule, it was true. He thought we must assume some higher influence than the cerebellum to explain the reflexes, and he believed that some function of the somæsthetic area was at work. This was very much in consonance with the theory promulgated by Dr. Onuf. He did not think any one to-day held that the motor tracts were absolutely and only motor. The facts of infantile cerebral palsy

show that defects of the somæsthetic area are not accompanied by paralysis, and it had been shown that the motor impulses starting from the cerebellum were modified in some way.

Dr. Starr queried if it would militate against Onuf's theory if it were considered that the pyramidal tracts were developed in animal life exactly in accordance with the development of the digital extremities. Dr. Spitzka has shown that in the seal, walrus and elephant the pyramidal tract is practically wanting, and develops in accordance with the motility of the digits. This fact was interesting in connection with Onuf's theory.

The president said that in spite of all the facts brought out this evening, the cases under consideration were, after all, exceptional. Granting that in the complete lesion of the cord above the lumbar level the knee-jerks were abolished, there were certainly a number of other cases, which, although the lesion was tolerably complete, the functions were not entirely lost. In Pott's paralysis it was a common thing for the knee-jerks to be exaggerated. He had had an autopsy in a case of dorsal Pott's, in which the cord was completely softened, yet the knee-jerks, instead of being lost, were exaggerated from beginning to end, and there were also contractures. For these reasons it did not seem to him wise to build up theories to explain exceptional cases. We would all be compelled to abandon the theory that the pyramidal tract conducted motion only, or chiefly. There was a fair foundation for the view that there was one cortico-spinal tract, and one cerebellar tract. After an examination of a large number of cases of spastic palsies in both adults and children, he had found very few cases in which there was no paralysis. He felt positive that these cases had more paralysis than spasticity or rigidity.

Dr. Onuf said that the pyramidal tracts had been found absolutely absent throughout the whole cord in a foetus of seven or eight months. This fact had been held to explain those cases of Little's disease in which there was no cerebral disease, and no paralysis, but only rigidity. It also tended to confirm the view that the pyramidal tracts were not strictly motor pathways.

Dr. Fraenkel, in closing, said that he did not doubt that any supra-lumbar disease of the cord would produce spastic paraplegia, but when there is a mechanical and pathological condition interfering progressively with the conducting function of the cord, and sufficient to give motor paralysis, the knee-jerks would be lost. Where there was a total anatomical destruction of the cord, the reflexes were abolished, probably because of the cutting off of the cerebral influence passing through the columns of Gowers. In a few recorded cases there was only partial destruction, yet the knee-jerks were lost as soon as the deep sensibility fibres were destroyed.

NOTES ON SOME ANATOMICAL CHANGES IN THE BRAIN CELLS
IN ACUTE ALCOHOLISM.

Dr. Charles L. Dana read a brief paper with this title, reporting ten cases, and the results of his studies in this direction in the past two years. He had used Nissl's stain for the most part. He stated that what was ordinarily known as acute alcoholic meningitis could not be said to be a meningitis at all, although clinically these persons died with all the symptoms of meningitis. The autopsy would reveal simple congestion and oedema of the brain, and even the microscope would rarely show any migration of leucocytes or anything approaching encephalitis. In some cases not even vascular activity would be observed. Alcoholic meningitis was not primarily a vascular disorder, but a slow poisoning; hence we must study the cell to determine the changes produced. It had been stated by some investigators that all forms of cell degeneration were the same, and that it was impossible, as Nissl claimed, to make out different cell degenerations in accordance with the particular pathological irritant. Whether this were so or not, certainly the microscopical appearances were different in the different cases of alcoholic meningitis, and in other cases associated with delirium and acute disorder prior to death. There was one type of degeneration quite characteristic of those dying from sunstroke, with intense delirium and very high fever. It consisted in a distinct and general pigmentation, involving the larger cells. This sudden development of pigmentary degeneration would seem to be characteristic of acute febrile degeneration, associated with acute toxæmia. Another kind of pigmentary cell degeneration was found in a case of pernicious anæmia. Here the pigmentation involved both the small and large cells. In a case of prolonged use of morphine and whiskey, in which death was due to exhaustion and malnutrition, the brain showed quite a general atrophy of both the nuclei and the cell bodies. There are three types of cell degeneration, viz.: (1) Intense pigmentation of the larger cells, chiefly, with degeneration of the cytoplasm; (2) a general cell atrophy of the body and nucleus; and (3) a good deal of change in the cell body, with many neuroglia nuclei in the peri-cellular spaces. In the cases of alcoholism and alcoholic meningitis it was not possible to make a distinct type of cell degeneration, nor could this hardly

be expected, as these patients die not so much from the alcohol as from auto-toxæmias and from the febrile process.

A CASE OF MENINGITIS SEROSA ACUTA.

Dr. C. A. Herter reported a case apparently coming in this category. The patient was a man, thirty-three years of age, who had enjoyed good health up to a year before his admission. While in the hospital he suddenly developed coarse clonic spasms; the feet were habitually in extension; there was slight clonic spasm in the flexors of the thigh. There was extreme and constant deviation of the head and eyes to the right. The spasms in the arms and legs were fairly symmetrical. The pupils were equal, of moderate size, and reacted fairly to light. The pulse was slow, high tension and fairly regular; the respirations were stertorous. On the following day the right arm was rigid and the left relaxed, and the coarse flexor spasm had disappeared. Then a hemiplegia developed on the left side; the pulse was rapid, feeble and of low tension. Towards the close of the day the patient could be aroused to partial consciousness. On the third day the irregular coarse spasms of the muscles began again, and were most marked at this time on the left side instead of on the right. The right pupil was dilated. On the fourth day the condition was essentially the same, but towards evening the irritative symptoms were less marked. On the fifth day all spasmodic movements ceased, and the patient remained comatose until death occurred. The temperature had been only moderately elevated. The autopsy was made by Dr. Ira Van Gieson. He found over the entire frontal and central lobes a thick layer of fluid between the pia and surface of the brain. The pia was milky, thickened and opaque. In the occipital and temporal regions this membrane was quite normal. The fluid was almost abundant in the frontal region, but was nearly uniformly distributed over the whole centre. The vessels and pia mater of the base were normal. The substance of the brain was pale, and the convolutions only slightly flattened. Microscopical examination showed the vessels of the pia to be largely obliterated. This effusion appeared to have occurred at the time of the onset of the acute symptoms. Probably the hemiplegia was due to pressure on the cortex. A sim-

ilar case had been reported in which the effusion had apparently been due to an infection.

Dr. Dana said that he had seen quite a number of cases of alcoholism presenting an almost identical pathological condition, although such a serous condition of the brain was not the rule in alcoholism. The clinical symptoms, however, did not permit of a differential diagnosis, nor did they indicate a sudden effusion.

Dr. Starr said that at operations on the brain a varying amount of fluid would be found under the pia, irrespective of the lesion present. Slight manipulation of the brain by the surgeon would cause a disappearance of this fluid. For this reason he did not think much stress should be laid upon similar pathological findings.

HEMORRHAGE INTO THE LATERAL VENTRICLES OF A NEWLY BORN INFANT.

Dr. Herter presented a specimen illustrating this condition. The child had been born at six months, and had lived in an incubator for a week. Labor had been difficult, but without forceps. A day or two before death extreme flexion of the toes and right index finger had been noted, together with a general and marked icterus. The autopsy showed nothing abnormal except in the brain. In the latter was a hemorrhage which accurately filled both ventricles.

The President remarked that hemorrhage into the brain had been reported as having occurred at some period of foetal life.

OCCIPITAL NEURALGIA FROM DISEASE OF THE COCCYX.

Oscar S. Brown (Med. Council, Sept., 1896) reports a case of severe occipital neuralgia, of four months' standing, cured by removal of a diseased coccyx. The author found that sudden pressure on the coccyx caused no local discomfort but sharp exacerbations of the neuralgic pain in the occipital region. A hypodermic needle removed a drop of fetid pus, and operation revealed necrosis of the entire coccyx, and about a dram of offensive pus. The operation was done several years ago, and the patient has not had "the slightest twinge of pain" since.

PATRICK (Chicago).